HAIR

Hair cycle:

- Autonomus , rhythmic transformation of fully developed hair follicle through phases of regression,growth,resting controlled by the follicle itself.
- Hair dose'nt grow in the same time ie ; There is Unsynchronization (some hair grow and some hair lost).

4 stages are known:

1.**Anagen** phase=active stage

2.**Catagen** phase (involuting stage) : involution of lower 1/3 of hair follicle by massive keratinocyte apoptosis

3.**telogen** phase=resting phase then new hair regrow

4.**exogen** phase=active hair shaft shedding(called anagen IV)(mostly vellus follicle)

• Total N of human hair follicles is 5000000. And there are 100000 in scalp

- 85%anagen - 0.5:1 % catagen -15%telogen.

• Duration of hair cycle for ex: scalp remember number 3

Anagen **3** years(2-6) Catagen **3** weeks(2-3) Telogen **3** months

N.B: Estrogen prolong anagen.

thyroxin promot growth.

corticosteroid retard anagen onset.

• **Hair growth:** 0.35 mm per day= 1 cm per month

N.B: Hair production is not increased by cutting or shaving.

Estrogen decrease hair growth rate.

Androgen increase hair growth rate ,hair diameter.

• Hair growth is mediated by Testosterone which converted by 5 alpha reductase to DHT,which promot conversion of terminal hair to vellus hair=hair loss

Finasterid , dutasterid are 5 alpha reductase inhibitors.

Hypertrichosis

Def : Increase the growth of body hair (Non androgen dependent) with normal androgen metabolism.

Hypertrichosis

Generalized Hypertrichosis:

1.Hypertrichosis Lanuginosa
2. Pigmented /terminal hair hypertrichosis

Hypertrichosis:

1.Congenital localized
2.Herditary hypertricosis
3-Localized Hypertrichosis
4-Acquired circumscribed H

Generalized Hypertrichosis

1-Hypertrichosis Lanuginosa

Congenital	Acquired
AR,fetal hair not replaced by vellus hair but Persist grow and may cover whole body	fine lanugo hair grow over large area of the body replacing normal hair, associated with para neoplastic syndrome and malignancy

2- Pigmented or terminal Hair hypertrichosis

Congenital generalized	Prepubertal	Drug-induced
* Generalized Hypertrichosis ±Gingival hyperplasia * Skeletal defect * Mental retardation * Other congenital anomalies * Maternal ingesion of minoxidil	1- Affect face(forehead pre auricular area) 2- Proximal extremities and back 3- Hair in the back in(inverted fire tree pattern)	Minoxidil D-penicillamine PUVA Streptomycin

Localized Hypertrichosis

1-Congenital localized	2-Hereditary Hypertrichosis
- Hamar Tomas (congenital melanocytic nevi—Becker"s) - Nevoid hypertrichosis - Faun tail: hypertrichosis in sacral area asign of spina bifida	Affecting the specific anatomic sites: - Hypertrichosis cubiti(hairy elbow syndrome) - Hairy palm,sole,auricle,eyebrow

3-Localized Hypertrichosis	4-Acquired circumscribed H
In heriditary and acquired diseases eg: Hypertrichosis with sun exposed areas>> is a sign of porpheria (PCT)	After trauma eg:surgical wound After inflammation due to prolonged CST use

D.D: with hirsutism.

Lab investigations: Normal androgen level.

Ttt of hypertrichosis:

1) Cosmotics: -
   hair bleaching of 6% H2O2
   shaving
   electrolysis ,thermolysis
   laser hair removal
   Intense pulsed light (IPL)
2) Stop using of drugs (minoxidile).
3) Ttt of underlying cause(malignancy).
4) Anti androgens if associated with elevated androgen level.

Hirsutism

Excessive growth of coarse ,terminal hair in women on androgen dependent areas of the body.

classification

1- Constitutional

2- Endocrine-organ based hirsutism

3- Iatrogenic

4- Ectopic Hormone production

5- Hepatic hirsutism

6- Failure of converting Androgento Estrogen

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1. Constitutional:- *Familial = Normal hormon level (end organ is more sensetive to androgen)

   *Androgen=Mild increase of DHEA-S(Di hydro epi andro steron sulfate

   *Ovarian=Normal DHEA-S + increase Free testosterone

   *Hyperprolactemia=increase prolactin H

2. Endocrine-organ H:- Adrenal:- thin female,central hirsutism.
   a) Non-tumoural adrenal H=adrenal hyperplasia &hypercortisolism,cushing syndrome
   b) Tumour adrenal H,Adenoma,Carcinoma

   Ovarian:- obese women = Menstrual disturbance,Lateral H.

   a) Non-tumaral PCO
   b) Tumour (ovarian T)

   Pituitary:- increase ACTH& prolactin

3. Ectopic Hormone production:- Lung carcinoma= ACTH secretion

   Chorio carcinoma= BHCG secretion

4. Latrogenic Hirsutism:- steroid administration
5. Hepatic hirsutism:- liver disease= decrease sex hormone binding globulin=increase testostero Free = increase DHT
6. Peripheral failure of conversion of androgen to estrogen:-lead to increase androstenedions

Management of Hirsutism

1. History:- age of onset,family history.menstrual and medical history
2. EX:- *Degree of H =Ferri man ,Gallwey score from 1-4(1)=no hair in chest,(4)=hair in chest,abdomen,back,thigh.

    * Other signs of virilism = acne,frontal balding, increase muscle man

    *CT,MRI,U.S of pelvis,abdomen to detect tumours

3.Lab investigations:-

*Total testosterone more than 200 nglml = androgenic tumours

*DHEA-S more than 700ngldl =adrenal tumour

*Increase 17- hydroxyl progesterone =congenital adrenal hyperplasia.

* PCO = increase testosterone,LH . LH:FSH = more than 3:1

*ACTH stimulation test :250 mg I.V infusion if more than 1000 SO,+ve test

*Dexamethasone suppression test.

4.TTT:-

1. Cosmotic: bleaching,shaving,laser,IPL
2. TTTof neoplastic causes: surgery,irradiation,chemotherapy
3. TTT of non neoplastic causes:--
    - Suppression of adrenal androgens:Dexamethasone 0.25-0.5 mglday for 3 months(Prednisolone 7-5 mglday for 2months then 5 mg lday for 2 months then 2 mglday For 6 months.
    - Suppression of ovarian androgens : OCP & GTRH antagonist for 6 months
    - Androgen receptor blockers: spironolactone 50-200 ngldayl6 months & cyproterone acetate (androcur) 20-100 mg from day 5 to day 14 of the cycle
    - 5 alpha reductase inhibitor like Finasteride 5 mg per day
    - Ketoconazol 400-1200 mg per day
    - Suppression op pituitary prolactin production (Cabergoline-Bromocriptine-dostenix)
    - Metformin in PCO
    - Topical therapy elforithine
    - Surgical for ovarian and adrenal tumours
    - Stop iatrogenic causes

Alopecia

Deff: Loss of hair from hairy regions of the body .

Evaluation of Alopecia

1- History 2- Examination 3-Investigations.

- History :
  1. Shedding or Thinin: Shedding hair falling is sever daily (TE, Anagen E).
     Thining of hair lead to visible scalp but without noticeble hair
     falling eg. AGA or Senile Alopecia.
  2. Duration : Congenital or acquired.
  3. Family History : hair shaft disorders , AGA.
  4. Hair care procedures :dyes , bleaching , cosmotics …
- Examination : patterned circumscribed (paten) eg. A.A

  1.Hair pattern &hair loss diffuse : T.E

  2. hair fragility due to hair shaft disorder : Trichoffexis redosa

  3.scalp surface . to see inflammation lead to alopecia

  eg. : Psoriasis , histiocytosis .

  4. Examination of other hairy region .

- Investigations :

  1.Gentle hair pull test : 50-60 hair grasped by forefinger and thumb then pull
  without causing pain. Normal < 10% of hair pulled are extracted
  (5) hairs or less . if more than 10% = T.E. The female shouldn't wash her hair 24-
  48 h before test

  2. Forcible hair pluck test (Trichogram) (hair root status)

Bundle of 50-60 hair is pulled out by artery forceps and the extracted hair is

examined under microscope. The female should be advised Not to wash her hair

5-7 days before the test.

Anagen hair : the root is the largest at its base .

The inner root sheath is present and firm.

Telogen hair : Club shaped rood , lack of angulation , loose sheath .

Dystrophic hair : pathological conditions . thin,without root sheath ,tapper at proximal end .

Normal Trichogram : The scalp contains 100,000 hair follicle

85% in anagen phase

0.5 -1% in catagen phase

15% in telogen phase .

Normally 25-100 hair (telogen) lost per day .

3-Tricho scan : Automated software program responsiple for analysis of hair growth .

4 -Time shed-hair count :

1. collecting hair loosed in 24 h (normally 50-100)

2. brush hair for 1 minute . count the number of hair shed hair normal (50) if >150 active

hair loss.

5-Hair growth window : For pt who say that their hair doesn't grow.

Shave area 2*2 cm after one week it should be 7* 0.35 mm =25 mm.

6-Scalp biopsy : to examine the presence of inflammation Or scarring two 4 mm punch biopsies are

examined.

7-Dermo scope

8- Lab investigations : CBC , S. iron , ….

For infection :Syphilis , KOH -→ Fungal

Cicatricial Alopecia

Occurs as a result of destruction of hair follicle by scar tissue leading to irreversible alopecia .

1ry : the hair follicle is the target of inflammation.

2ry : Follicles are destroyed in a non specific way (burn – traw)

Causes of Cicatricial Alopecia :

1- Congenital : Ictyosis (recessive x-linked).

Darier 's disease - epidermd nevi.

Epidermolysis bullosa (Recessive dystrophy).

2-Acquired :

1. Physical Injury : mechanical trauma , burn , radiation
2. Infection → Fungal kerion , favus & viral ; varicella , HZ

   Bact. : LV , qumma , leprosy ,carbuncle , acne nerotica

   Protozoa : lishmaniasis
3. Tumours BCC , SCC , metastatic carcinoma .
4. CTCL
5. Collagen disease DLE , morphea ,DM.
6. Dermatosis of unknown etiology : LP - sareoidosis

   Cicatricial pemphigoid – Acne keloidalis

   Pseaudo follicuiltis barbe – Pyoderma gangernosum.
7. Clinical syndromes : pseaudo pelade & borcq folliculitis decalvins.

Pseaudo pelade of brocq woman to man 3:1

Condition in which there is chronic patchs of alopecia which are slowly progressive , irregular defined , affecting the crown or back of the head .

early stage : patches with mild perifollicular erythema.

Late stage : patch smooth shinny atrophy without sign of inflammation and abscence of follicular pores . DIF -ve or minind IgM at BMZ of infundibulum.

H/P : moderate perifollucular infiltrate (lymphocytic) around upper ½ of follicle later .

the epidermis (thin ,atrophic) dermis is (sclerotic) .

Collagen bundles run vertically to the skin mark follicle / erector pilli in place

Classification of 1ry Cicatricial Alopecia

	Scalp	Treatment
Lymphocytic 1-DLE	Symptomatic, erythematus scalp plaques with follicular plugs telangectasia,atrophy depigmentation with time. Activity in the centre of alopecia patch.	IL corticosteroid+ Topical cs. Hydroquinone,prednisolone, Tacrolimus,Tazarotene,sotretinoin.
2-Lichen plano pilaris: a) Classic	Pruritic, multifocal or central alopecia patches with follicular hyperkeratosis,erythema at hair bearing margin.	What mentioned above + Cyclosporine Griseofulvin Antiandrogens(Finasteride&dutasteride)
b) Frontal fibrosing Alopecia	Frontotemporal recession of hair at hair bearing margin,eye lash loss,facial papules and body hair involvement in sever form.	
c) Pseudoplade of Borcq	Asymptomatic,non inflammed ivory-white or flesh coloured small oral round confetti-like reticular or large irregular patches + or- atrophy.	Topical cst IL TA Prednsolone Hydroquinone Isotretinoin
<u>Neutrophilic:</u> Folliculitis Decalvans	More in central scalp,grouped follicular pustules, Military abscess or hair bearing margin.	Abcs +steroids Rifampicin+ 2nd Abcs Fucidic acid +Zinc
<u>Mixed:</u> Acne Keloidalis	Occipital scalp (nape) firm red-brown papules,	IL TCA + OR – Antibiotics Excision of plaque form.

	papulopastules,nodules,Keloid plaques.	

Telogen Effluvium

Def : Acondition of diffuse hair loss in which the anagen hair follicles enter into telogen phase Prematurly leading to diffuse shedding . Usually occures after sever stress

Causes:

1. Physiology :- physiological effluvium of new born& post partum effluvium.
2. Stress or injury:- *High fever(Malaria) *Sever infection(HIV,Syphilis)
   *Ch disease(SLE,liver,renal) *Hypothyroidism
   *Crash aliquid protien diet *Malnutrition
   *Iron deficiency anaemia *Zinc defeciency
   *Major surgery *Drugs &Retinoids
   *Heavy meals *B.blockers
   *Hormones,Antithyroids,Anticonvulsant, Anticoagulant (heparin)

Acute TE:- diffuse shedding of hair occurs 3-4 months after stress exposure . It continous for few months and regrowth take place within 6 months with good prognosis.

Chronic TE :- telogen hair shedding is longer than 6 months

Pathogenesis:- 1. Immediate anagen release: Follicles enter telogen phase prematurely= Acute TE.

2.Delay anagen release : in pregnancy, hair follicle remain in anagen Phase and post partum it convert to telogen . result in increase shedding =TE=Post partum hair loss.

3.Short anagen syndrome:shortening of anagen phase So, club hair are Released 4-6 week after the onset of anagen =AGA &Chronic idiopathic TE.

4.Immediate telogen release: Shortening of normal telogen due to drugs Like Minoxidil.

5.Delay telogen release :Prolonged telogen followed by transition to Anagen. In some humans.

Diagnosis:

History: - Duration of shedding & hair care procedures.

History of surgery,fever stress,pregnancy.

History of chronic illness liver,renal,malignancy.

Menstrual history,diet history

Examination:

1. Inspection (manual-woods light,dermoscope).
2. Inflammation : erythema,scaling,infection.
3. Hair collection,hair pull,hair pluck,hair clipping
4. NAIL examination.

Lab investigation: CBC ,, CMP ,,S.iron ,, S.zinc ,, TSH ,, T4 ,, Vit.D.

TTT: of the underlying disease +nutritional suplement +Minoxidil 2% or 5%.

Androgenic Alopecia

Def: progressive transformation of terminal follicles into vellous follicles.It is induced by androgen stimulation of geneticaly predisposed persons

Pathogenesis:

1. Genetic AD ,the inheritance is polygenic. Female has less family history.
2. Androgens : there is relation bet AGA and androgens castrated male before puberty don't develop baldness but they develop baldness if they receive testosteron
3. Aging : baldness is progressive by aging.

Diagnosis:

1. Family history (maternal ,paternal).
2. Trichogram and scalp biobsy :

* Normal total number of follicles with increase proportion of telogen hair.

*Reduction of the size of hair follicle = decrease diameter.

Terminal hair : Vellous hair reduce (more than 8:1) to (less than 4:1).

Anagen : Telogen ratio reduce from (12:1) to (5:1).

3.lab test : for androgen increase in female if manifestation of hyper androgenism is present

Clinical features:

Male: Start with bitemporal recesion followed by balding of vertex,sparing the post,lateral scalp margins.

Female: Start in late life ,not sever as male .its usually diffused than patterned(thining) affecting frontovertical area but unlike men,the frontal hair line retains .widening of central part shows christmas tree pattern. Grading according to central part widening(Ludwing)

Grade 1=minimal widening , Grade 2=moderate , Grade 3=significant widwning +thining

TTT:

1. Systemic ANDROGENS: Finastride = 1% for males (FDA approved) and 2.5% for females (not FDA approved).
2. Topical : Minoxidil = 2% FDA approved for male and females ,5% approved for males only.
   Combination with tretinoinenhance the trichogenic effect og minoxidil
   Vasodilator= increase cutanous bl flow to scalp and increase conversation of vellus hair to terminal hair in 305 of patients.
3. Light therapy:increase vascular circulation,ATP production ,enhance matrix cell proliferation 665 nml red light(hairmax-laser comb)
4. Cosmotic =hair transplantation.
5. Prostaglandin analogues: Topical Latanoprost 0.1 % increase hair density.
   Injection of bimatoprost 0.03% weekly for 12 week is good for female pattern hair loss.

Alopecia Areata

DEFF: Common skin disorder, Non scaring alopecia present as circular area of alopecia which can lead to total scalp hair loss = Alopecia totalis OR complete scalp and body hair loss =Alopecia universalis.

Etiology:

1. Genetic :
    - 20% have +ve family history.
    - Associated with congenital disease(Down"s syndrome).
    - Associated with Atopy.
    - Associated with HLA DQ7,DQ3,DR11.
2. Immunological:
    - AA is an autoimmune disease. Auto-antigen is most propaply melanocyte associated antigen.
    - Associated with other autoimmune disease (LP,vitiligo).
3. Emotional stress.
4. Hormonal fluctuation ,infection,vaccines are triggering factors of AA .

Histopathology:

1. Presence of miniature hair structure of early anagen or telogen
2. Peri bulbar lymphocytic infiltrate (SWARM OF BEES) mainly T .helper type CD4 to CD8 is higher indicate activity .

Ther is 2 stages :

a) Early stage (progressive): MN cell infiltrate around bulb + hair matrix changes +trichomalacia +narrowing of hair shaft.
b) Stable disease : Scanty peribulbar infiltrate arrested anagen hair.

Clinical features:

Sudden and complete hair loss in acircumscribed area in which the skin is completely normal.

Exclamation mark hair: Found around the patch which indicated progression.

The hair shaft is thin proximally and thick distally ,that can be easly pulled out.

Ophiasis: Extension of AA along the scalp margin (Active).

Alopecia totalis: total scalp hair loss.

Alopecia universalis: total scalp and body hair loss.

AA diffusa: AA associated with diffuse hair loss as TE.

Reticular AA: recurrent patchy disease.

Sites: Scalp ,, beard ,,moustache,,eye brow.

Trichogram : Normal or telogen hair root But if progressive rapidly = telogen dystrophic.

Associations: مهمه جدا

1. Atopy
2. Eye changes = cataract with Alopecia totalis.
3. Nail changes: 20% of cases . Pitting + longitudinal ridging +thickening the intensity which parallel of hair loss.

Course and prognosis is variable

Bad prognosis in: مهمه جدا

1. Atopy
2. Ophiasis
3. Multiple lesions
4. Presence of exclamation mark.
5. Affection of eye brow
6. Nail changes.

TTT of AA:

*Topical:

1. Topical or intracutaneous CST Kenacort.
2. Non specific irritant Tincture Iodine, Tr capsicum.
3. Photochemotherapy PUVA.
4. Anthralin.
5. RPP
6. Minoxidil.
7. Prostaglandine analogues Latanoprost, bimatoprost.

*Systemic :

1. Systemic CST . only in rapid progressive active AA. Prednisolone 0.5-0.8 mg per kg per day slowly traped over 2 months.
2. Systemic cyclosporine.
3. Methotrexate
4. Biologics (infliximab- eta nercept) but failed.

Drug induced alopecia

Diffuse non scarring reversible alopecia in scalp.

Mechanism:

1. Direct effect on hair follicle by :

- Interruption of anagen growth (anagen effluvium) like,Chemotherapy ,anti cancerous and Colchicine.
- Androgenic induction of normal terminal hair into villous hair (androgenic progesterone,Anabolic steroid ,exogenous androgen).

- Premature peri ceptitation of telogen (T.E).

2. Indirect: drug induce systemic disorder leading to hair loss
    - Hypothyroidism
    - Lichenoid drug

    Both lead to cicatricial alopecia or TEN.

Trichotillomania

Psychological disorder. The ptn has habit of twisting the hair around his finger and pull it out.

Femal : male =5:1 & child : adult =7:1 & boys more than girls.

C .P: ill-defined area (patch) in which the hair is twisted and brokenof various distance of normal skin scalp.

Sites : eye brow , eye lash , pubic hair.

Pathogenesis : Impulse control disorder causing sever stress or impairment.

Criteria of Trichotillomania:

1. recurrent self hair pulling out result in hair loss.
2. increase tention immediately befor pulling out the hair.
3. pleasure or relief sensation after pulling out the hair

Trichogram in affected area: decrease telogen hair +2ry trichodystrophies with fracture distal end . Un affected area is Normal.

Growth window : Steady increase in hair density in the shaved area.

Histopathology :

- Distorted follicular anatomy without inflammation.
- Multiple catagen follicles.
- Pigment casts and keratin plugs (Trichomalacia).

D.D: مهمه جدا

- Scaly ringworm : Trichotillomania (no scaling & -ve fungal culture).
- Aopecia areata : in Trichotillomania (no Exclamation mark).

TTT: Minor or Major tranquilizer (SSRI).